of lassitude, headaches, and constipation for about two months. There was no family history of tuberculosis, and no signs or symptoms of this disease could be discovered in the child. A radiograph of the lungs showed nothing abnormal. He had never suffered from "bilious attacks," and was an only child. As will be seen from the accompanying chart, the temperature became

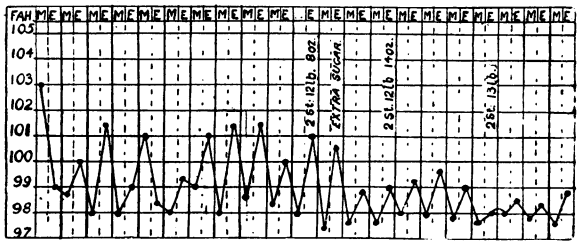

Chart showing subsidence of prolonged unexplained pyrexia on administration of sugar.

normal almost immediately after giving extra sugar (3 drachms of glucose powder in lemonade three times a day). It will be seen also that he at once began to gain in weight. Since then he has attended as an out-patient at intervals, and now (October, 1928) he is apparently in normal health, having had no further symptoms. During the whole of this period he has continued to take glucose.

The following conclusions would appear to be justified.

1. Cyclical vomiting is an extremely common affection in children of all classes.

2. It is due to depletion of the glycogen reserves of the liver.

3. It occurs mainly in "nervous" children, attacks usually being precipitated by mental stress.

4. It can generally be prevented by giving extra sugar in the diet.

5. The child's desire for sweets subserves a physiological function, and should be gratified.

6. A large proportion of cases of "debility" is due also to sugar shortage.

7. Such debility can generally be cured and prevented by increasing the allowance of sugar in the diet.

8. Sugar shortage is the most common cause of headaches, constipation, abdominal pains, and "growing pains" in children.

POSTSCRIPT.

Since writing the above I have read a paper on "The pre-rheumatic child," by Professor C. W. Vining of Leeds (Bath Conference on Rheumatic Disorders, 1928). In it he shows that a very large proportion of frankly rheumatic children give a preceding history of various symptoms of minor ill health, lasting for months or years. He groups these symptoms under the heading of "toxic debility," and shows that their incidence is very much less in normal children. This state of "toxic debility," Professor Vining suggests, is intimately and causally related to the incidence of acute rheumatic fever, and he concludes that "the success of our efforts to prevent and control rheumatism (acute) will be in proportion to the amount of recognition we give to the pre-rheumatic health defect, and I would suggest on the evidence brought forward in this paper that prevention lies first and foremost in the provision of a well-balanced diet from infancy onwards." The symptoms Professor Vining includes under the title of the "pre-rheumatic" state are identical with those I have suggested as being due to a relative insufficiency of sugar in the diet. As, moreover, they may be readily cured and prevented in the majority of cases by the addition of sugar to the diet, it is possible that this dietetic modification affords a means of preventing acute rheumatism, or at least of diminishing its frequency.

REFERENCES.

[1] Frew, R. S.: *Lancet*, 1911, ii, 1264. [2] Marfan: *Arch. de Méd. d'Enf.*, xxiv, January, 1921. [3] Gamble, Ross, and Tisdall: *Transactions* Thirty-fourth Annual Meeting American Pediatric Society. [4] Ross, S. G., and Josephs, H. W.: *Amer. Journ. Dis. Child.*, xxviii, 447, 1924. [5] Brown, M. J., and Graham, G.: *Arch. Dis. Child.*, i, 302, 1926. [6] Hilliger: *Jahrb. f. Kinderheilk.*, lxxx, 1, 1914.

SUDDEN DEATH FROM PNEUMONIA WITHOUT APPARENT SYMPTOMS.

BY

JAMES A. STEPHEN, M.B., D.P.H.,

CHILD WELFARE MEDICAL OFFICER, ABERDEEN;

AND

EDWARD R. C. WALKER, M.B., M.R.C.P.ED.

SUDDEN deaths in marasmic infants in the first year of life are not uncommon, especially in the presence of severe anhydraemia. Heart failure, respiratory failure, or what appears to be a sort of general nervous collapse, sometimes brought on by an unsuitable meal, may extinguish suddenly the flickering little life. When the child seems to be doing well and death occurs without any warning symptoms the matter becomes very puzzling. Such deaths are commonly certified as being due to "marasmus," a term which is inadequate, if not actually inaccurate. The following case of this nature, therefore, seems to us of some importance and interest.

A female infant, born on November 13th, 1927, was admitted to the Burnside Infant Welfare Home, Aberdeen, on December 1st, 1927, weighing 6 lb. 9 oz. She was put on the routine feed for infants of her age—namely, lactic acid milk sweetened with dextri-maltose—but did not gain weight satisfactorily. Little could be made out clinically, except that after a few weeks a pustular eruption developed on the chin. This cleared up fairly readily with a coal-tar and zinc paste. There was no albuminuria. Progress, however, remained unsatisfactory, the gain being only 26 ounces in fifteen weeks. At this point one unit of insulin daily was given subcutaneously, followed by a drachm of glucose by the mouth, a method of treatment which has given some happy results in our hands. In the first week of this treatment the child gained 11 ounces, and in the next 3 ounces, and in every way appeared to be much improved. At 2.20 a.m. on March 25th she was found in her cot just dying. During the week prior to death the temperature, taken morning and evening, varied between 97° and 98.2° F., the pulse between 124 and 130, and the respirations between 22 and 28. The 10 p.m. feed on March 24th was finished as usual; at 1.45 a.m. on March 25th her napkins were changed, and there was no suspicion that anything was amiss. The last dose of insulin was given at 3 p.m. the previous day, nearly twelve hours before death.

At the *post-mortem* examination the lower lobes of both lungs were found to be solid and in the red hepatization stage of lobar pneumonia, microscopical sections showing the alveoli to be filled with red cells and leucocytes. A pure culture of pneumococci was grown from the affected lung substance. In the upper lobes were found scattered patches of broncho-pneumonia. The only other abnormality was a large thymus. The thyroid did not appear to be abnormal in any way. Unfortunately, these glands were not weighed.

Discussion.

The point of immediate interest in connexion with this case is that a sudden, unexpected, and apparently inexplicable death in an infant in the first year of life may be due to pneumonia. Dr. Simonds, in Abt's *Pediatrics* (vol. 8, p. 630), indeed, states that broncho-pneumonia is the most common cause of sudden death in the first year of life. Lobar pneumonia is not mentioned in this connexion. While lobar pneumonia is much less frequent in children under 1 year, and is held by some not to occur at all, our experience, confirmed by clinical, radiological, bacteriological, and pathological findings, is that it does occur, and in the case here recorded the pathological and bacteriological findings were as stated above. Discussing the mechanism of death in these cases, Simonds considers that the fatal result is brought about by toxic absorption from the focus, and, probably chiefly, by asphyxia induced by the great reduction in alveoli, which are small in infancy, available for blood exchange. In our case, if asphyxia were the chief cause of death, it would be necessary to postulate its very sudden onset, for it is difficult to see how a child with any degree of respiratory embarrassment could, without showing some recognizable signs of distress, take a feed of several ounces as this child did four hours before death, at which time the pneumonia must almost certainly have been fairly well established. An overwhelming toxaemia seems to be the more probable explanation. In reviewing the case from birth to necropsy four points stand out—namely, (*a*) the failure to gain weight normally on an adequate diet, arguing a faulty metabolism; (*b*) increase in weight in response to insulin;

(*c*) complete lack of resistance to infection, to the extent of failure to produce symptoms; and (*d*) the presence of a large thymus gland. The sequence of cause and effect may have been as follows. An innate weakness was present of endocrine outfit in particular, probably of thyroid, pituitary, and adrenals, indicated by the response to insulin, which we believe exerts its beneficial effect indirectly through stimulation of pituitary and thyroid; and possibly also by the presence of a large thymus. On account of this deficiency metabolism was faulty and gain in weight was unsatisfactory. Stimulation of the so-called "katabolic" glands by insulin brought about increased metabolic activity with consequent gain in weight. A pneumococcal infection occurred, and the endocrines, probably innately deficient, or perhaps weakened by stimulation, failed along with the autonomic nervous system to respond with defensive activity and consequent production of symptoms, and death occurred. This explanation is in line with that suggested by Ramsay in his interesting paper on "The resistance factor in disease" in the *Journal* of April 14th, 1928 (p. 628).

If this interpretation be approximately true, then it seems possible that another class of case may be rescued from that limbo of infantile diagnosis termed variously "marasmus," "wasting," "debility," and "athrepsia"; and, if this be a correct conception of the pathology of these cases, therapeusis based on it should hold out hopes of their more successful treatment. The rational procedure would appear to be to supplement the deficient glands by giving reliable glandular extracts, and concurrently, or probably later, to stimulate them by some such method as insulin injections.

Summary.

A case of sudden death in an infant of 4 months, from pneumonia which produced no symptoms, is reported. It is suggested that these cases are commoner than is supposed, and also that the underlying pathology is concerned with an innately deficient endocrine system. Treatment based on the latter supposition is proposed.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

SPREADING SUBCUTANEOUS EMPHYSEMA AFTER FRACTURE OF RIB.

THE following case is of special interest since it illustrates an effective treatment of spreading subcutaneous emphysema following the fracture of a rib; two instances of this condition were recently published in the *British Medical Journal* by Mr. J. A. C. Macewen (November 17th, 1928, p. 892). The patient in the present case left the wards of the Albert Dock Hospital towards the end of December.

A dock labourer, aged 54, was admitted on October 5th, 1928, after having fallen 15 feet into the hold of a ship, together with a heavy wooden hatch, which fell across his chest. There was fracture of the seventh left rib in the posterior axillary line, with a small patch of emphysema over the site of fracture. On the succeeding days the emphysema spread rapidly as follows: October 5th: A circular patch about 6 inches in diameter. October 6th: Left scapular and both pectoral regions. October 8th: Left axilla and upper arm, left supraclavicular fossa. October 9th, 10 a.m.: Chest wall emphysematous throughout, with spread to the left side of the abdominal wall, the left arm down to the wrist, and the right arm down to the elbow. 6 p.m.: Emphysema covered both arms to the wrists, the entire surface of the body, and both legs to the knees. There was a sudden spread to the neck and face, up to the supraorbital ridges; the whole face was now uniformly spherical with the eyelids ballooned and standing out from the general level, giving an appearance not unlike that of two golf-balls on the surface of a football. 7 p.m.: "Eggshell" crackling could be felt in the finger-tips and nail beds, and respiration was becoming so seriously embarrassed that tracheotomy was contemplated. Acting on a suggestion by Mr. T. Pomfret Kilner, surgeon to the hospital, pleural puncture by a medium-sized trocar and cannula was performed on the affected side, permitting free ingress and egress of air, with partial collapse of the lung.

The theory advanced for this treatment was that the continuous increase of emphysema was due to escape of air by way of the pleural cavity into the subcutaneous tissues under pressure; if, then air were allowed to pass freely out of the pleural space this pressure would cease to operate.

There was no further spread of the emphysema, and after twelve hours the cannula was removed, respiration being less embarrassed. Recovery was uneventful, the emphysema passing off in the reverse order of its appearance; on October 25th there was none present.

It would appear from this experience that so simple an expedient as pleural puncture at an opportune moment would avert the necessity for the accepted treatment by multiple incisions in situations where the subcutaneous tissues are distended.

Albert Dock Hospital, London, E. ROBERT V. GOODLIFFE, M.R.C.S., L.R.C.P.

AN UNUSUAL CASE OF HYPERKERATOSIS BLENNORRHAGICA.

CERTAIN uncommon characteristics would seem to render this case of hyperkeratosis blennorrhagica worthy of record.

A patient contracted gonorrhoea in August, 1924; he was ill for seven days, and had no complications. He was reinfected in December, 1927; there were no complications, and he was discharged cured after forty days' treatment. He was readmitted to the Royal Naval Hospital, Haslar, with gonorrhoea on March 1st, 1928; there were no complications, and he was discharged cured on April 9th.

The patient was readmitted to the Royal Naval Hospital, Haslar, on August 24th with well-marked hyperkeratosis blennorrhagica of both feet only. He stated that two months prior to admission to hospital he noticed dried skin appearing in different places, first on the right heel, secondly on the left heel, then spreading over the soles and toes, and becoming gradually more painful on pressure, especially while standing on guard. He finally was put on light duty on account of inability to perform his work.

It is stated by most authorities that the following three symptoms constitute the syndrome: gonorrhoeal urethritis, gonococcal arthritis, and hyperkeratosis. The point of interest is that the above patient has never had any form of joint trouble whatsoever since his first infection with the gonococcus in 1924, and no joint trouble has been present up to the time of his discharge "cured" from hospital on September 26th. Secondly, the patient had not noticed any urethral discharge prior to readmission to hospital on August 24th, and none has been present since.

Examination of a urethral smear after prostatic massage on August 27th, 1928, revealed a few Gram-positive organisms, together with an occasional pus and epithelial cell. Urine cultures on August 28th and September 11th were sterile, and a urethral smear on September 10th was negative. A urethroscope examination was negative, and the prostate was normal. The patient was a fit, healthy man in every other respect.

We have had four cases of hyperkeratosis in this hospital during the last two years; three patients had urethritis, and arthritis in conjunction.

In the present case hyperkeratosis was the only symptom, and probably the infection of March, 1928, could be held responsible for this. The point of interest is that if such a disease as gonorrhoeal iritis can occur ten years or more after initial infection, and appear as the only metastatic exhibition, is it not possible that hyperkeratosis may appear as the sole manifestation also?

The treatment applied was purely local—namely, alkaline foot baths and nikalgin ointment.

T. R. LLOYD JONES,
Royal Naval Hospital, Haslar. Surgeon Commander, R.N.

INJECTION TREATMENT OF VARICOSE VEINS.

I RECENTLY had a difficult case of varicose veins to deal with, and the satisfactory sequel to a change of procedure may justify its publication.

The internal saphenous was very bad, with nine large nodules from the internal condyle upwards, the lower leg being like a fisherman's net with veins of varying sizes. I began with a 0.5 c.cm. dose quinine urethane, and for the space of five weeks increased the dose until I gave three 1 c.cm. injections at the one sitting. The first injections were given at the upper end of the vein, and the later ones at the lower; all were given slowly and cautiously, and none had any effect. I decided, therefore, to adopt different tactics.

I thoroughly prepared the nodule at the internal condyle, emptied it, and kept it empty by digital pressure. I then partially dilated it by injecting forcibly 2.5 c.cm. of quinine urethane. This was kept in the nodule for thirty seconds and then the leg was lowered for thirty seconds, the patient standing on it; after this the leg was elevated for one minute, and then the patient was instructed to walk about. The result was sclerosis of the meshwork on the leg and of the nodule with two inches of the vein above it and of one other nodule. A week later I gave a similar injection half-way up the thigh, and after another week had elapsed found no further injection necessary.

The reasons for bringing this case to notice are the large single dose employed; the forceful injection; and finally, the injection in the middle of the vein, which ensured that either centrifugal or centripetal flow would be of equal advantage.

Benbecula, South Uist. S. BARTLETT.